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Background on Health Effects of Acid Aerosols

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This introduction to the 1987 NIEHS-EPA Symposium on the Health Effects of Acid Aerosols reviews the state of our knowledge on this topic as of the close of the 1984 NIEHS Conference on the Health Effects of Acid Precipitation (*Environmental Health Perspectives*, Volume 63) and the results of some key studies completed since that time. These studies, together with the results of the studies presented in the papers that follow, provide a substantial increment in our knowledge of the health effects of acid aerosols.

Introduction

This Symposium on the Health Effects of Acid Aerosols is taking place 3 years after the NIEHS Conference on Health Effects of Acid Precipitation. The earlier conference, also convened at the NIEHS, covered the effects of acid precipitation on mobilization, transport, and speciation of toxic metals, the impact of acid precipitation on metals toxicity, and the health effects of inhaled acid aerosols. Its proceedings were published in *Environmental Health Perspectives*, Volume 63. This symposium was organized to help the sponsors (NIEHS and EPA), the larger scientific community, and Congress address and assess the expanding data base on the effects of inhaled acid aerosols.

To the extent that we can place the emerging exposure and effects data into a broader perspective on the public health consequences of current and future exposures, we can help the sponsors identify data gaps, research needs, and the possible need for a primary national ambient air quality standard for acid aerosols. To this end, we depend on the skills and insights of the rapporteurs of our half-day sessions on Exposure, Epidemiology, Toxicology, and Acute Human Responses to present integrations and interpretations of the presentations in their sessions. We will also benefit from an evaluation of the potential uses and limitations of the current data base on acidic aerosols for risk assessment by Roy E. Albert (*1*). His broad perspectives, combined with his experience in experimental studies on the effects of inhaled irritants, environmental epidemiology, and quantitative risk assessment, enable him to evaluate the data base from a broad perspective.

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Historical Background

Industrial pollution from steel mills and sulfuric acid (H_2SO_4) plants were implicated in excess deaths and respiratory illness among elderly people in The Meuse Valley in Belgium in December 1930, and in Donora, PA, in October 1948. Coal smoke containing H_2SO_4 was implicated in excess deaths, primarily in elderly people in a series of episodes in London from 1952 to 1962, with approximately 4000 excess deaths from the December 1952 episode alone. During that period, deaths attributable to chronic bronchitis were higher in London than in other, less polluted areas in the U.K., and much higher than in other countries in northern Europe. Aggravation of bronchitis among patients in London with chronic lung disease was also demonstrated to occur as pollution levels increased. Unfortunately, levels of exposure to H_2SO_4 were not determined in the era when excessive mortality and morbidity were clearly evident, and exposure-response relationships were made, if at all, with pollutant indices referred to as British Smoke (BS) and sulfur dioxide (SO_2). The actual measures were the blackness of sampled particles (BS) and the net acidity of sampled vapor (SO_2).

It is now clear that excess mortality in London was much more closely associated with BS than with SO_2. However, BS is primarily a measure of the carbon content of the aerosol, an unlikely causal factor. It seems much more likely that another aerosol constituent, e.g., the free hydrogen ion (H^+), was a causal factor.

Many of the reasons for the interest in and concern about H^+, or H_2SO_4 as one dominant source of ambient H^+, were discussed at the 1984 NIEHS conference. My assignment at that conference was rapporteur for the session on the health effects of inhaled acid aerosols. Table 1 from the proceedings of that conference

indicates the questions I had formulated prior to the conference. It also presents the answers I could come up with by the end of the conference, the conditions under which they were applicable, and the remaining knowledge gaps at that time. My responses were based, in large measure, on material presented at the conference, but were supplemented, as appropriate, with knowledge of other relevant work (*2*).

Recent Progress

Three years is not a long time for progress in research on so broad and complex an issue as the health effects of acid aerosols, especially when there have been so few active investigators and resources available to address the knowledge gaps. Most of the active research groups are represented at this symposium and will be presenting their recent work to us. It is therefore too early to fully gauge the progress that has been made, although much of it is significant and moves our knowledge base well past that of 3 years ago, as summarized in Table 1. Most of the recent research provides exposure-response data involving directly measured acid aerosol exposures. However, we are not yet at the stage where we can afford to ignore studies with surrogate measures of exposure. Reports from Germany and Poland at this symposium are good examples of papers of great interest that still rely on surrogate metrics of exposure.

Other recent work not being reported at this sym-

Table 1. Summary of major issues concerning exposures to airborne acidity and their human health effects.

Question	Answer[a]	Applicable conditions	Knowledge gap
Have health effects from exposure to ambient acidic aerosols been demonstrated in the past?	Yes	~600 cases morbidity; primary H_2SO_4; point source avg. exposure ≈ 160 $\mu g/m^3$	Diagnostic criteria; exposure variability in time and space
Do current North American exposures to ambient acidic aerosols produce measurable health effects?	Uncertain, but likely	Asthma admissions, secondary aerosol	H^+ vs. surrogate SO_4^{2-}
		Wheeze, primary plume aerosol	H^+ vs. surrogate SO_2
		Excess daily mortality, secondary aerosol	H^+ vs. surrogate SO_4^{2-}
What are current North American exposures to ambient acidic aerosols?	Max. ~30 $\mu g/m^3$ as H_2SO_4 (6 hr avg.)	Summer, rural N.E. U.S.	Spatial, diurnal, and seasonal variations
What are the effects of single brief exposures to acidic aerosols on respiratory mechanics?	Transient bronchoconstriction in exercising asthmatics	100 $\mu g/m^3$ H_2SO_4, adolescents 350 $\mu g/m^3$ H_2SO_4, adults	Effects in more severely disabled patients
	Persistent reduction in DL_{CO} in guinea pigs	< 40 $\mu g/m^3$ H_2SO_4	Significance to human health effects
What are the effects of single brief exposures to acidic aerosols on particle clearance from the lungs?	↑ -mucociliary clearance	≤ 100 $\mu g/m^3$ H_2SO_4, humans ≤ 250 $\mu g/m^3$ H_2SO_4, rabbits	Effects on lung epithelium
	↓ -mucociliary clearance	> 500 $\mu g/m^3$ H_2SO_4, humans > 750 $\mu g/m^3$ H_2SO_4, rabbits	
	↑ -alveolar clearance	1000 $\mu g/m^3$ H_2SO_4, rabbits	
	↑ -activation of macrophages	1000 $\mu g/m^3$ H_2SO_4, rabbits	
What are the effects of repetitive exposures to acidic aerosols on lung structure and function?	↑ secretory cells ↑ epithelial thickness ↑ alveolar clearance	250-500 $\mu g/m^3$, 1 hr/day, 5 day/week, rabbits	Progression of epithelial changes
What are the implications of persistent structural and functional alterations in the lung to the pathogenesis of chronic respiratory disease?	T-B tree: unknown, of concern because early changes parallel those from cigarette smokers	Repetitive daily exposure to irritants, stimulation of mucus secretion	Effects on respiratory mechanics and symptoms at later stages
	Alveolar region: unknown, of concern because persistent inflammation can lead to abnormal repair processes	Continuous mild inflammatory response	Effects of macrophage activation and protracted recruitment of inflammatory cells and fibroblasts; structural changes in deep lung
Are there especially sensitive subgroups in the population? If so, who are they?	Yes: asthmatics, especially adolescents and with vigorous exercise	Bronchoconstriction, 100 $\mu g/m^3$ H_2SO_4 or 0.3 ppm NO_2	Effects in more severely disabled patients
Do other ambient pollutants potentiate the effects of acidic aerosols on the respiratory tract?	Yes		Other mixtures, sequences of exposures

[a]↑ an acceleration in rate; ↓ a retardation in rate.

Table 2. Growth-adjusted pulmonary function differences in 7- to 11-year-old school children during and after an air pollution episode in Ijmond, the Netherlands, January 16-20, 1985 (3).

Lung function parameter	Differences compared to baseline values of Nov.-Dec. 1984, (± SE)		
	January 18 (n = 62)	February 6 (n = 60)	February 15 (n = 41)
FVC, mL	−62 (± 11)[a]	−58 (± 13)[a]	−2 (± 16)
$FEV_{0.5}$, mL	−39 (± 9)[a]	−30 (± 13)[b]	−5 (± 13)
FEV_1, mL	−50 (± 10)[a]	−43 (± 13)[a]	7 (± 15)
PEF, mL/s	−219 (± 62)[a]	−69 (± 76)	−162 (± 68)[a]
$MEF_{75\%}$, mL/sec	−100 (± 67)[c]	−50 (± 70)	−36 (± 58)
$MEF_{50\%}$, mL/sec	−37 (± 44)	−70 (± 48)[c]	7 (± 47)
$MEF_{25\%}$, mL/sec	−27 (± 24)	−32 (± 34)	26 (± 38)
MMEF, mL/sec	−38 (± 32)	−60 (± 44)[c]	12 (± 40)

[a] $p < 0.01$ (one-sided).
[b] $p < 0.05$ (one-sided).
[c] $p < 0.1$ (one-sided).

posium should also be noted. In particular, I would like to call your attention to papers by Dassen et al. (3) and Ozkaynak and Thurston (4).

Dassen et al. (3) measured pulmonary function in primary school children in the Netherlands before, during, and after the January 1985 acidic pollution event that caused elevated exposures throughout northwestern Europe. Reports of studies examining population responses in England and West Germany will be presented in Session II of this Symposium. During this episode, the Dutch children were exposed to respirable particulate and SO_2 concentrations in the range of 200 to 250 $\mu g/m^3$; their baseline functions were measured about 4 to 6 weeks earlier when the pollutants were below 100 $\mu g/m^3$. During the episode, function indices were significantly lower by 3 to 5%, and decrements were still present at 16 days later, but not at 25 days. Some of the results are summarized in Table 2.

Ozkaynak and Thurston (4) reported on associations between 1980 U.S. mortality rates in 98 Standard

Table 3. Summary of Ozkaynak and Thurston's results from regression model for 98 U.S. SMSAs and for 38 SMSA subset with directly measured FP and IP concentrations using the 1980 total annual mortality and ambient particle data.[a]

Pollution variable	Coefficient (SE) for pollution variable, deaths/10^5 persons per year per $\mu g/m^3$	
	98 SMSA	38 SMSA
SO_4^{2-}	6.6 (1.5)‡	6.9 (2.1)†
FP[b]	2.2 (0.8)†	2.8 (1.4)*
IP[c]	0.3 (0.3)	0.3 (0.5)
TSP	0.1 (0.2)	0.1 (0.3)
FP	—	2.9 (2.1)
IP	—	1.0 (0.9)

[a] Model includes the variables percent over 65 years, median age, log of population density, percent with 4 years of college, and percent below poverty line.
[b] Estimates of FP based on regional FP equations developed by Ozkaynak and Thurston (4).
[c] Estimates of IP based on approach developed by Trijonis (6).
*$p < 0.05$.
†$p < 0.01$.
‡$p < 0.001$.

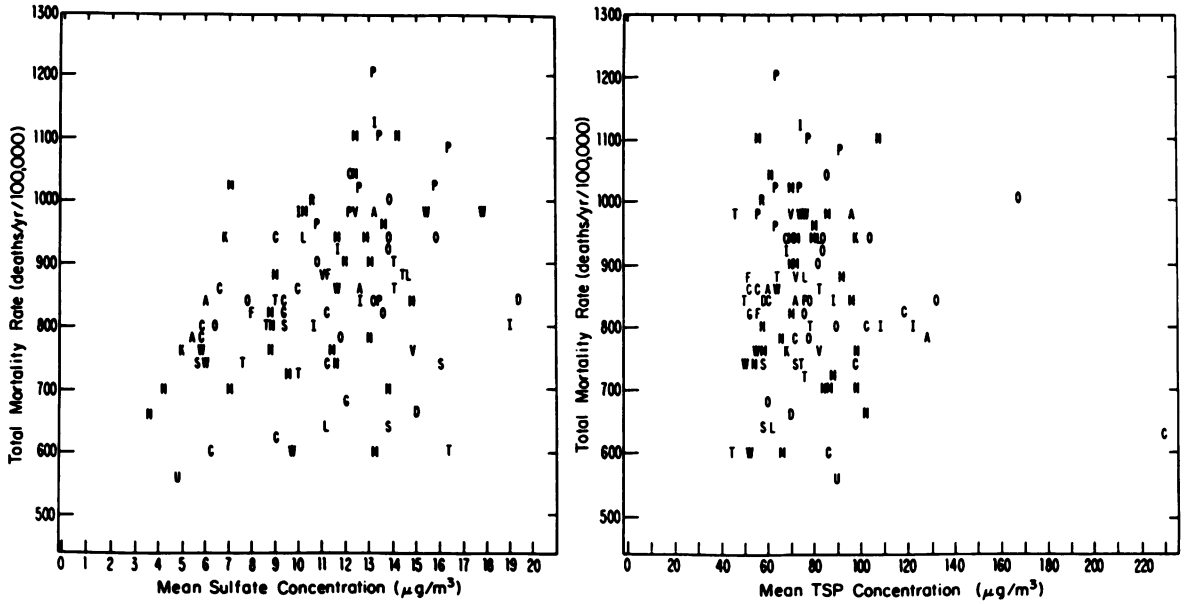

FIGURE 1. Plot of total mortality rate versus annual mean sulfate concentration (left panel) and total suspended particulate matter (TSP) concentration (right panel) in the 98-SMSA subset. (Letters are initials of states.) Adapted from Ozkaynak and Thurston (4).

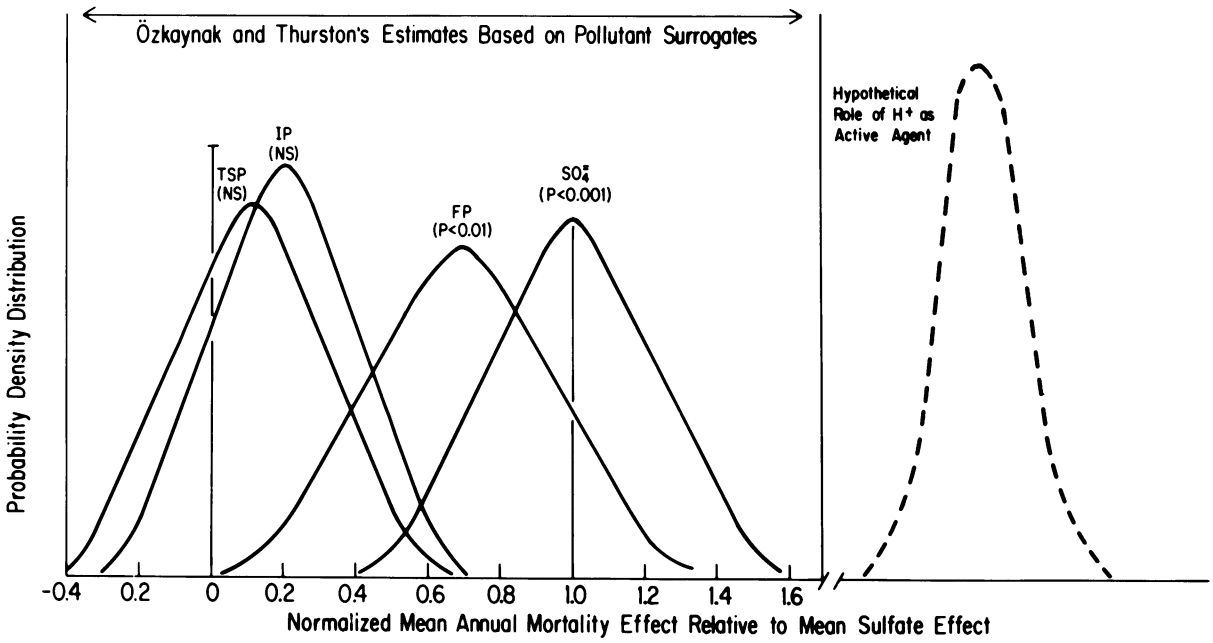

FIGURE 2. Comparative estimates of mean mortality effect of chronic exposure to TSP, IP, FP, and SO_4^{2-} particle concentration measures for 98 SMSA model of Ozkaynak and Thurston (*4*) and hypothetical effect of aerosol H^+ concentration.

Metropolitan Statistical Areas (SMSAs) and four measures of particulate air pollution. These were total suspended particulate matter (TSP); inhalable particulate matter, i.e., particulate < 15 μm in aerodynamic median diameter (IHP); fine particulate matter, i.e., particulate < 2.5 μm in aerodynamic median diameter (FP); and sulfate (SO_4^{2-}), a major component of FP. This was a final report on the preliminary analyses reported by Ozkaynak and Spengler (*5*) at the 1984 NIEHS Conference. They found that FP and SO_4^{2-} were most consistently and significantly associated with the reported SMSA-specific total annual mortality rates, whereas TSP and IP were often nonsignificant predictors of mortality. These results are summarized in Table 3 and Figure 1. Figure 2 is my adaptation of the results of the SMSA-mortality analyses, showing a hy-pothetical extrapolation of the analytical results based upon the hypothesis that the acid component of the particulate, a greater fraction as one goes from TSP to IP to FP to SO_4^{2-}, is the causal factor. The results reported at this symposium provide a basis for examining the validity of this hypothesis, and/or for helping to establish a better one.

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